Electrolytes, Orthopaedics & Elderly Care: Hyponatraemia Revisited.

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Introduction

Electrolyte imbalance in the elderly is a clinical problem faced by both elderly-care physicians and orthopaedic surgeons[1]. The abnormalities in homeostatic mechanisms that manifest with age can have dramatic consequences for the unwary clinician. The issue of per-operative fluid management in the elderly was raised by Lane and Allen in a 1999 BMJ editorial following an incident of iatrogenic hyponatraemia[2]. This review focused on the attention given to the per-operative electrolyte management of elderly women with hip fractures and highlighted the importance of prompt diagnosis and treatment.

In light of this paper by Lane and Allen, we wanted to establish the incidence of hyponatraemia within our orthopaedic population. We also aimed to determine whether this was different from the general elderly population by establishing a control group of elderly care patients for comparison.

Methods

We undertook a retrospective, consecutive analysis of the serum sodium concentrations of all patients admitted to the trauma ward of a teaching hospital with a fractured neck of femur during a three-month period. The biochemical profile on admission and subsequent serum electrolyte levels were recorded for these patients. The serum sodium levels were observed in each patient for either two weeks following admission or, where the inpatient stay was less than this, until discharge. For the purpose of comparison with the admission sodium the mean serum sodium from the samples taken postoperatively was calculated. In addition the fluid administration regime was observed and recorded for each patient. For the purpose of analysis we chose to define hyponatraemia as a serum sodium level below 135mmol/L.

An age-matched control group of patients admitted to the elderly care ward over the same period were similarly observed and used for comparison with the orthopaedic patients. The data were analysed statistically within each patient group using a paired t-test and between groups using an independent t-test.

Results

Two hundred patients were identified, 100 hip fracture patients and 100 elderly care patients. Results from all of the patients were used for analysis with no loss to follow up. The mean age of the study group was 81.3 years (Table 1) with no difference between the two groups ($p>0.05$).

	n	Mean Age (years)	Admission sodium (mmol/L)	Mean FU sodium (mmol/L)	Mean change in sodium (mmol/L)
All patients	200	81.3	135.7	136.6	0.9 (95% C.I 0.3-1.4)
Hip fracture	100	80.2	135.5	136.4	0.9 (95% C.I 0.2-1.6)
Elderly care	100	82.5	136.0	136.8	0.8 (95% C.I 0.0-1.6)

Table 1: Changes in the serum sodium during the course of admission in the hip fracture patients, elderly care patients and the group as a whole.

The mean admission serum sodium of all patients studied was 135.7mmol/L (SD=5.4). The results for the two individual groups are given in table 1. There was no significant difference between either admission or follow up sodium levels between either group - admission sodium p=0.49, follow up sodium p=0.52.

In both groups the sodium level rose by just under 1mmol/L during the course of the admission (Table 1). In all groups this increase was statistically significant ($p<0.05$). There was no statistically significant difference between the observed changes in serum sodium between the hip fracture and elderly care groups (p=0.82). This seems to indicate that over the period of observation the two groups behaved similarly.

While the mean serum sodium levels were greater than 135 mmol/L in all groups the actual percentage of cases presenting to hospital with hyponatraemia was 29% in the hip fracture group and 33% in the elderly care group. We also noted that of those patients that were hyponatraemic on admission, the majority remained hyponatraemic during their hospital stay (23 of 29 hip fracture patients and 20 of 33 elderly care patients). Relatively few patients with 'normal' sodium on admission developed hyponatraemia whilst in hospital (3 hip fracture patients and 4 elderly care patients).

Every hip fracture patient was administered at least 1 litre of 0.9% saline immediately on admission. This was in keeping with the department's protocol for the management of hip fractures. On average, each patient received 1.5 litres of normal saline prior to surgery but other fluids including Hartmann's solution and 0.18%/4% dextrose saline were occasionally also administered. These were administered to one third of the patients along with the normal saline.

Discussion

The homeostatic mechanisms associated with water metabolism are vulnerable to age-related degeneration and to the various disease processes and medical interventions that frequently befall the elderly[3]. Although predominantly an extracellular cation, serum sodium flux has a marked impact on water movement throughout the compartments. The serum sodium concentration can be seen as a balance between extracellular fluid volume and extracellular sodium content. Abnormalities in this balance can arise from an excess of extracellular water, a deficit of extracellular sodium or at worst, a combination of both. Hyponatraemia reflects an abnormal ratio of sodium to water and is defined as a serum sodium concentration of less than 135 mEq/L.

Within our study hyponatraemia was a common finding in the elderly population being admitted to hospital with both orthopaedic and medical conditions. The incidence in our study was higher than the 15% reported in earlier literature[4] and was seen in approximately a third of all admissions. This is perhaps due to the fact that the groups we looked at were more elderly with a mean age of over 80.

We used a random elderly care population as a control group for comparison with our orthopaedic hip fracture patients to look for differences between them and the general elderly population admitted to hospital (Figure 1). The admission sodium level for both the hip fracture and elderly care patients was very similar, and no significant difference was found between these two groups. Likewise there was no significant difference in the observed hyponatraemia between the two populations throughout their care in hospital. This seems to demonstrate that hyponatraemia is not a condition confined to orthopaedic patients, as the elderly care patients were affected and behaved similarly.

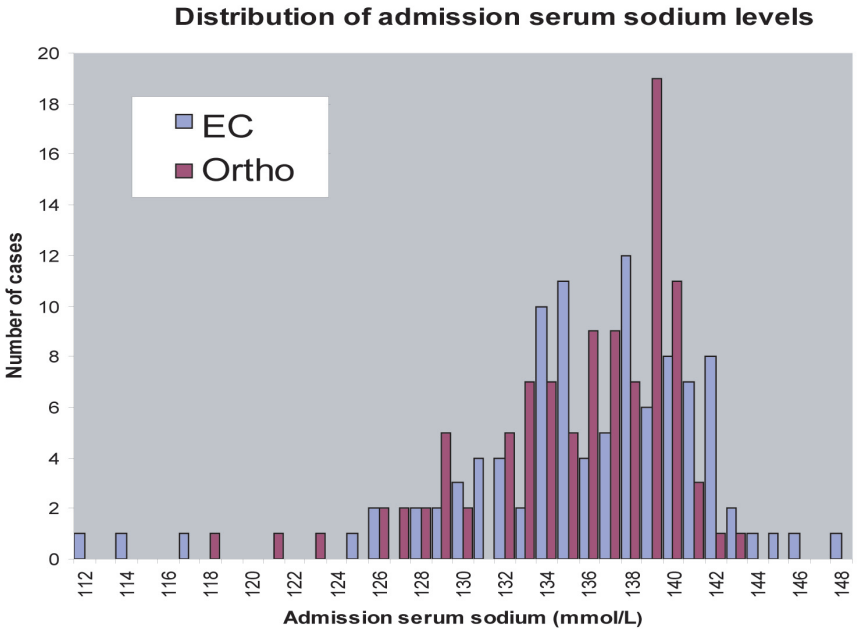

Figure 1: Distribution of the admission serum sodium levels for the elderly care (EC) and hip fracture (Ortho) groups

It was those patients found to be hyponatraemic on admission that continued with low sodium levels for the duration of their treatment in hospital. This was true for both the orthopaedic and the elderly care population. Very few patients admitted with a normal sodium level became hyponatraemic during their stay. The current hip fracture protocol used within our unit states that all patients admitted with hip fractures are given at least 1 litre of normal saline in the first 24 hours following admission. There is also an increased awareness among junior doctors of the dangers of prescribing sodium-free fluid regimes following the publication of articles like Lane and Allen's. It is possibly for these reasons that so few patients became hyponatraemic during admission.

Of the large number of patients admitted with hyponatraemia only a relatively small number were corrected into the normal range after two weeks or at the time of discharge. The patients in the orthopaedic group were however treated to get their serum sodium to a level which was deemed safe for surgery by the anaesthetist. While this absolute value varied between anaesthetists, the value that was usually accepted was a serum sodium greater than 130mmol/L.

Conclusion

This study underlines the high incidence of hyponatraemia within the elderly population. It also demonstrates that there is no difference in the incidence of hyponatraemia between the elderly orthopaedic population and the general elderly population. It demonstrates the importance of correct fluid management and a high clinical suspicion amongst clinicians for prompt investigation of the sometimes innocuous symptoms that may herald the catastrophic and avoidable consequences of this condition.

Summary of the management of Hyponatraemia

Hyponatraemia is a condition frequently encountered among the elderly population and is seen in around 15% of patients admitted to hospital overall[4]. In the elderly it often results from retention of water secondary to impairment of free water excretion. Occasionally it is due to sodium loss exceeding that of water – this is associated in particular with the use of thiazide diuretics and elderly patients may be especially susceptible to this condition[5].

The causes of hyponatraemia are numerous but can be thought of as occurring in association with either a decreased, normal or increased extracellular volume[6] (Table 2). Clinicians must also be wary of factitious hyponatraemia, which is usually due to a "drip arm" sample taken from a limb being infused with a hypotonic solution or laboratory error.

The symptoms associated with hyponatraemia are largely non-specific. Severity of symptoms is related to the extent of the hyponatraemia and the rapidity of onset. They include increasing weakness, lethargy and nausea. Late manifestations are seen with altered conscious level and fitting. These late symptoms in particular are associated with cerebral alterations in tonicity and cell swelling. The vague nature of the symptoms accounts for the frequent delay in diagnosis and lack of robust management plan in these patients[7].

The initial management should be directed towards establishing the severity, chronicity, rapidity of onset and possible underlying cause. Key steps include careful history and examination and particular attention should be made to drug history and chronic health conditions. Diagnosis is aided by biochemical analysis of urinary and serum sodium and osmolality levels, and serum cortisol levels[8].

To enable clarity in managing the hyponatraemic patient, it is useful to break these cases into two distinct groups: 1) the hypervolaemic patient and 2) the euvolaemic or hypovolaemic patient as this gives some indication of the possible underlying aetiology (see Table1). In salt-deficient patients treatment is aimed at replacing lost sodium whereas in patients with water excess fluid restriction and review of diuretic therapy is more appropriate.

Of equal importance is establishing the rate of onset of the hyponatraemic state, as over-zealous correction of chronic hyponatraemia is potentially harmful. An acute change can be safely rectified rapidly but if the hyponatraemia has developed slowly the brain will have adapted by decreasing the intracellular osmolality. In these cases a rapid increase in extracellular osmolality will result in severe shrinkage of brain cells and the syndrome of central pontine myelinolysis with often irreversible symptoms becoming manifest around a week after correction[9].

In hyponatraemia of more chronic onset, identification and treatment of the underlying cause is paramount. Adequate management of contributing disease or simply withholding exacerbating drug therapy may be enough to return the patient to an improved biochemical profile[10]. If the above measures are insufficient, a fluid restriction of 1–1.5 litres daily can be used. In all cases, the fundamental step is the identification and prompt treatment of any underlying lesion.

In acute severe hyponatraemia (serum sodium <120mmol/l) prompt treatment is necessary as there is a high risk of cerebral oedema and hyponatraemic encephalopathy[11]. Management of this severe acute onset condition is by cautious administration of hypertonic saline, not exceeding 2 mmol/l per hour and avoiding normal sodium levels for the first 48 hours[12,13].

Decreased Extracellular volume - Usually due to sodium loss in excess of water leading to sodium deficiency. *Usually due to excessive sodium loss from the gut (vomiting, diarrhoea, bleeding) or kidney (osmotic diuresis due to hyperglycaemia, diuretics, Addison's disease, tubointestinal renal disease)*
Normal Extracellular volume – Usually related to abnormalities in the release or action of ADH. *Abnormal ADH release, SIADH, increased ADH sensitivity, action of ADH like substances, psychogenic polydipsia.*
Increased Extracellular volume – Usually due to intake of water in excess of the kidneys ability to excrete it. *Seen predominantly in patients with cardiac failure, hepatic cirrhosis or nephrotic syndrome.*

Table 2: Causes of Hyponatraemia.

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